Desaturation delay, parameter for evaluating severity of sleep disordered breathing

A. Kulkas[1], P. Tiihonen[2], P. Julkunen[2], E. Mervaala[2], and J. Töyräs[2,3]

[1] Department of Clinical Neurophysiology, Seinäjoki Central Hospital, Seinäjoki, Finland
[2] Department of Clinical Neurophysiology, Kuopio University Hospital, Kuopio, Finland
[3] Department of Applied Physics, University of Eastern Finland, Kuopio, Finland

Abstract— **Sleep apnea hypopnea syndrome (SAHS) is considered as a major health problem causing increased risk of cardiovascular problems. One of the most often used parameters to assess the severity of SAHS is apnea hypopnea index (AHI). It is calculated as the average amount of total cessations of breathing (apnea events) and partial reductions in breathing (hypopnea events) per hour of sleep. AHI only takes into account the total number of events regardless of their true nature. In this paper ambulatory polygraphic recordings of 19 male patients were analysed and the blood oxygen desaturation delay (BODD) studied in estimating the cardiovascular stress associated with sleep disordered breathing. The delay of the desaturation event occurring after apnea and hypopnea events were studied. Correlation with AHI and the delay parameter is modest and it shows variation between patients with similar AHI. This suggests that the delay parameter may provide additional information about the probably varying cardiovascular stress in patients with similar AHI and in the diagnostics of sleep disordered breathing in general.**

Keywords— **Sleep disordered breathing, Apnea, Hypopnea, Blood oxygen desaturation delay, AHI**

I. Introduction

Sleep apnea hypopnea syndrome (SAHS) is characterised by excessive daytime sleepiness [1]. It is a complex public health problem with estimated prevalence of 2% in adult women and 4% in adult men [2]. Many researchers, however, believe that the true prevalence of the syndrome is heavily underestimated [3,4]. Apnea and hypopnea events are defined as cessation or reduction (<50%) of breathing for more than ten seconds [1,5]. Hypopnea needs to be also associated with blood oxygen desaturation or arousal event [1,5]. SAHS can cause cardiovascular problems [6,7] besides the impairment of daytime functions and it is also a risk factor for mortality [7,8]. Early diagnosis and estimation of the severity of sleep disordered breathing is vital to ensure treatment and prevention of worsening of the disease and related health problems.

Today the gold standard method for diagnosis SAHS is polysomnography (PSG). In polysomnography breathing is estimated by using several sensor measuring number of physiological signals [1,5]. From these physiological signals various parameters can be computed based on the visual or automated analysis of the data. Polygraphy (PG) is a lighter version of PSG that includes all the physiological signals related to respiration of PSG excluding electroencephalography (EEG) measurements.

One of the most commonly used parameter for evaluating the severity of the syndrome is the Apnea-Hypopnea index (AHI) [1,5]. It is defined as the average number of apnea and hypopnea events per hour during the sleeping period [1,5]. Besides AHI sub-indexes such as apnea index (AI) containing the average number of apnea events per hour and hypopnea index (HI) containing the average number of hypopnea events per hour are used [1,5]. Besides the breathing events oxygen saturation drops are also commonly evaluated [1,5]. Oxygen desaturation index (ODI) contains the average number of desaturation events per each hour of the sleeping period [1,5]. It must be noted that AHI, AI, HI and ODI take into account only the average number of the apnea and hypopnea events or the average number of desaturation events occuring during the hours of sleep [1,5] regardless of the duration of the events or the nature of the saturation drop related to the event.

There are four different severity categories related to AHI. Normal AHI ≤ 5, mild $5 < \text{AHI} \leq 15$, moderate $15 < \text{AHI} \leq 30$, and severe $30 \leq \text{AHI}$. If SAHS remains untreated it has tendency to progress into more severe categories [9-11].

Commercially available PSG systems provide reports of the physiological signals measured. Typically these reports include mean and maximum values of e.g. the event durations of apnea, hypopnea and desaturation events. There are recommendations that suggest that parameters such as number of hypopnea and apnea events and minimum oxygen saturation value should be reported [5].

The parameters currently recommended for diagnostics of SAHS are focused on calculating the number of events [5] and do not take into account in more details the nature of the events or the relation between the desaturation and apnea and hypopnea events. In both cases after apneas and hypopneas the following desaturation can start at different times in relation to the start of apnea and hypopnea events. The delay of the desaturation following apnea and hypopnea events could provide valuable information about the ability

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of the physiological system to compensate against sleep disordered breathing. This way one would gain additional information about the cardiovascular stress related to SAHS. When considering the limitations of traditional AHI, which takes into account only the average amount of the events per hour of sleep, the search for additional, more appropriate information is justified. This raises also the question whether AHI is the best tool for evaluating the patient status and prognosis.

In the present paper, potential of the use of blood oxygen desaturation delay (BODD) in the diagnostics of SAHS and sleep disordered breathing in general was studied.

II. Patients and Methods

A. Recordings

In the present study 19 ambulatory polygraphy recordings of male patients were analysed retrospectively. The median age of the patients is 53 years (range 36 – 73) and the median BMI 27.2 kg m^{-2} (range 22.0 – 34.3). The study was approved by the local ethical committee of Kuopio University Hospital. Patients were referred to Kuopio University Hospital due to sleep problems and they were given the polygraphy recordings device and written instructions to perform the recordings at home.

Table 1. Patient demographics.

Patient #	AHI (1/h)	Age (years)	Weight (kg)	Height (m)	BMI (kg/m^2)	Disease severity
1	2.6	55	68	1.76	22.0	NoSAHS
2	3.0	56	80	1.73	26.7	NoSAHS
3	3.2	47	87	1.83	26.0	NoSAHS
4	3.0	46	87	1.80	26.9	NoSAHS
5	9.9	59	85	1.78	26.8	Mild
6	9.9	40	72	1.79	22.5	Mild
7	10.1	54	78	1.75	25.5	Mild
8	10.7	58	85	1.77	27.1	Mild
9	21.1	52	93	1.85	27.2	Moderate
10	22.2	49	98	1.72	33.1	Moderate
11	22.1	36	79	1.67	28.3	Moderate
12	22.1	56	94	1.79	29.3	Moderate
13	42.0	56	102	1.88	28.9	Severe
14	41.8	49	88	1.76	28.4	Severe
15	51.5	73	70	1.70	24.2	Severe
16	51.2	53	95	1.75	31.0	Severe
17	60.1	53	90	1.67	32.3	Severe
18	70.0	39	105	1.75	34.3	Severe
19	71.1	58	94	1.68	33.3	Severe

Figure 1. Example of the blood oxygen desaturation delay (BODD) calculated after hypopnea and apnea events. The window length is 150s. The delay is calculated as the time between the start of desaturation and apnea or hypopnea events. The curves from the top: thermistor, blood oxygen saturation, abdominal respiratory movements, and body position.

For this study patients with similar AHI were randomly selected from each AHI severity category. From the three AHI severity categories (normal, mild, moderate) four patients with similar AHI were randomly. As the range is wider in the severe class than in the milder ones seven patients with different AHI were randomly selected from the severe category. The demographics of the patients are presented in table 1.

The polygraphic recordings were performed with *Unisalkku* device (Neurotech Oy, Kortejoki, Finland) and consisted of four channels, thermistor for measuring the breathing ariflow, abdominal respiratory movements, blood oxygen saturation (Minolta Pulsox-7, Japan) by a finger pulse oximeter and body position sensor. All the signals were sampled at 4 Hz frequency.

B. Analysis

The recordings were analysed by an experienced medical physicist according to the standard respiratory rules [5]. For hypopnea scoring hypopnea rule 4A was used [5]. Each hypopnea event scored was followed by a desaturation event.

A desaturation event was also linked to apnea event if the beginning of desaturation event was inside 60s window from the beginning of the apnea event. The delay from the beginning of hypopnea event to the beginning of the related desaturation event and the delay from the beginning of apnea event to the beginning of the related desaturation event were calculated.

An example of hypopnea and apnea events and the related desaturations and the calculated BODD is presented in figure 1.

The average blood oxygen desaturation delay related to hypopnea and apnea events and combined BODD related to both hypopnea and apnea events were then determined for each patient. The combined (apnea and hypopnea) average blood oxygen desaturation delay was correlated with AHI.

III. Results

The mean BODD after hypopnea events (n=1352) and after apnea events (n=2210) were 25.8s and 24.5s, respectively. Based on the present results the blood oxygen desaturation delays after both hypopneas and apneas show significant patient to patient variation independent of AHI (Figure 2.). The combined BODD (after both apneas and hypopneas) shows also inter patient variation and the variation does not directly follow the severity classes of AHI (R^2=23, n=19, Figure 3).

IV. Discussion

The traditional parameters such as AHI and ODI take into account the average number of events per hour occurring during sleep regardless of the nature of the events. This raises a question whether these commonly used parameters are the best tools for evaluating the severity of sleep disordered breathing.

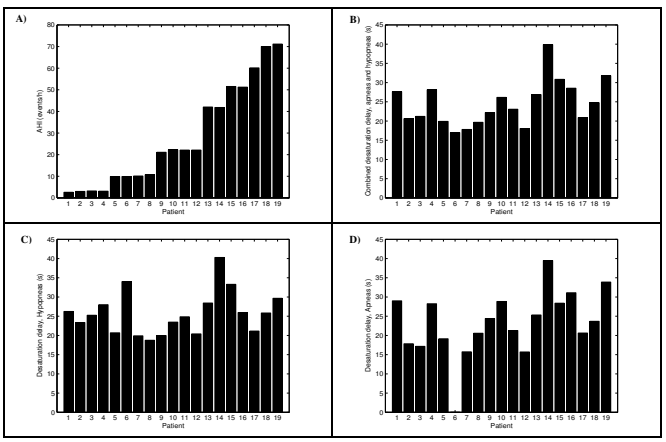

Figure 2. Significant patient to patient variation in the blood oxygen desaturation delay independent of AHI. Panel A) AHI, panel B) average desaturation delay related to both hypopneas and apneas, panel C) average desaturation delay related to hypopneas, and panel D) average desaturation delay related to apneas. Patient number six did not have apnea events during the recording.

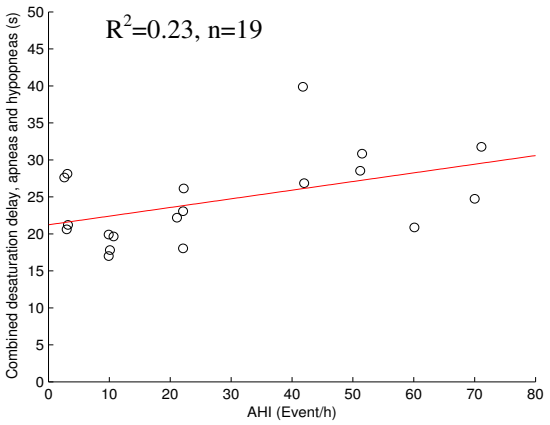

Figure 3. Traditional AHI and combined (apnea and hypopnea) blood oxygen desaturation delay correlation. The correlation, denoted as R^2, is modest, which suggest that desaturation delay can provide new information about the severity and consequences of sleep disordered breathing.

In the current paper blood oxygen desaturation delay, BODD, parameter was calculated and evaluated. Desaturation delay was calculated for both apnea and hypopnea events separately and a combined delay for both events was also calculated and evaluated. In principle the BODD parameter should provide information of the functionality sympathetic nervous system and the related cardiovascular stress and therefore valuable information in estimating the severity of sleep disordered breathing and the need of treatment of each individual.

The BODD may reflect the physiological consequences of the hypopnea and apnea events and also the ability of the physiological system to compensate against these breathing abnormalities during sleep. Each individual may have different properties in terms of coping with the breathing abnormalities during sleep.

The blood oxygen desaturation delay showed inter-patient variation independent of AHI. The moderate correlation with AHI may suggest that within patients with similar AHI there is inter subject variation in the related cardiovascular stress. Therefore the consequences of sleep disordered breathing may be different even with patients with similar average number of apnea and hypopnea events during hours of sleep.

It is well known that sleep disordered breathing is related to cardiovascular problems and has relation also to the mortality of the patients [6,12]. With more detailed analysis of the sleep disordered breathing events the cardiovascular stress might be estimated in a new manner and the BODD parameter could bring new insights to the diagnostics and prognosis of sleep disordered breathing.

It seems that the delay parameter presented here should be further evaluated along with AHI in follow up studies of patients suffering from sleep disordered breathing.

Sleep disordered breathing is a complex public health problem with increasing prevalence [3,13,14]. It is known that untreated SAHS has tendency to worsen over the years [9-11]. Therefore early diagnosis of sleep disordered breathing and novel means for estimating the true severity and need for treatment are needed to prevent the increasing risk of cardiovascular problems and mortality related to sleep disordered breathing.

V. Conclusions

Sleep apnea hypopnea syndrome is a complex public health problem with constantly increasing prevalence. Traditionally the severity of the disease is estimated with parameters such as apnea hypopnea index which takes into account the average number of events occurring during the hours of sleep. These kinds of parameters have limitations as the true nature of the events is often discarded. Also the relation of desaturation and hypopnea and apnea events is not investigated in detail. Blood oxygen desaturation delay, BODD, parameter consisting of the delay between breathing cessation events and partial breathing reduction (apnea and hypopnea) and desaturation events was studied here. The moderate correlation with AHI and inter patient variation of the novel parameter with patients with similar AHI suggests that there could be different cardiovascular stress related to sleep disordered breathing patients with similar AHI. The desaturation delay parameter might bring new insight in the diagnostics of sleep disordered breathing and the related cardiovascular stress.

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References

1. American Academy of Sleep Medicine (1999) Sleep-Related Breathing Disorders in Adults: Recommendations for Syndrome Definition and Measurement Techniques in Clinical Research, Sleep 22:667-689
2. Young T, Palta M, Dempsey J et al. (1993) The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 328:1230-1235
3. Gibson GJ (2004) Obstructive sleep apnoea syndrome: underestimated and undertreated. Br Med Bull 72:49-64
4. Skomro RP, Kryger MH (1999) Clinical presentations of obstructive sleep apnea syndrome. Prog Cardiovasc Dis 41:331-340
5. Iber C, Ancoli-Israel S, Chesson AL et al. (2007) The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology, and Technical Specifications. Westchester, Ill: American Academy of Sleep Medicine
6. Roux F, D'Ambrosio C, Mohsenin V. (2000) Sleep-related breathing disorders and cardiovascular disease. Am J Med 108:396-402.
7. Young T, Finn L, Peppard PE et al. (2008) Sleep disordered breathing and mortality: eighteen-year follow-up of the Wisconsin 15 sleep cohort. Sleep 31:1071-1078.
8. Punjabi NM, Caffo BS, Goodwin JL et al. (2009) Sleepdisordered breathing and mortality: a prospective cohort study. PLoS Med 6:40 e1000132.
9. Sahlman J, Miettinen K, Peuhkurinen K et al. (2010) The activation of the inflammatory cytokines in overweight patients with mild obstructive sleep apnoea. J Sleep Res 19:341-348.
10. Tishler PV, Larkin EK, Schluchter MD et al. (2003) Incidence of sleep-disordered breathing in an urban adult population: the relative importance of risk factors in the 10 development of sleep-disordered breathing. Jama 289:2230-2237.
11. Young T, Peppard PE, Gottlieb DJ (2002) Epidemiology of obstructive sleep apnea: a population 20 health perspective. Am J Respir Crit Care Med 165:1217-1239.
12. Marin JM, Carrizo SJ, Vicente E et al. (2005) Long-term cardiovascular outcomes in men 25 with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. Lancet 365:1046-1053.
13. Peter JH, Koehler U, Grote L et al. (1995) Manifestations and consequences of obstructive sleep apnoea. Eur Respir J 8:1572-1583
14. Wittmann V, Rodenstein DO (2004) Health care costs and the sleep apnea syndrome. Sleep Med Rev 8:269-279

Author: Antti Kulkas
Institute: Department of Clinical Neurophysiology, Seinäjoki Central Hospital, The hospital district of South Ostrobothnia
Street: Hanneksenrinne 7
City: Seinäjoki
Country: Finland
Email: antti.kulkas@epshp.fi